INVITED COMMENTARY

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The high-risk patient has been haunting us for years. Before the endovascular era, very few patients were actually denied open aneurysm repair on account of extensive cardiovascular comorbidity and the increased mortality this type of surgery would be associated with. With the advent of endovascular aneurysm repair (EVAR), things changed. Because this therapy was initially considered most appropriate for patients who had a prohibitive risk for open surgery, an epidemic of high-risk patients seemed to ensue. In the subsequent years, many efforts were made to define "high risk," but a widely accepted definition could not be accomplished.

Recently, the EVAR-2 trial generated renewed interest in the preoperative risk profile of abdominal aortic aneurysm (AAA) patients, as it concluded that EVAR offers no benefit for high-risk patients compared with best medical treatment. Unfortunately, the EVAR-2 trialists had also failed to agree upon a clear definition of their high-risk patients.

The Glasgow Aneurysm Scale (GAS) is a simple score that can be calculated in the office without complex formulas. It provides a numerical score representing the preoperative risk profile of an AAA patient. In a retrospective study, Hirzalla et al aimed at an external validation of the GAS score as a predictor for postoperative mortality and major morbidity after elective open AAA repair. They conclude that the GAS is a valid predictor for postoperative mortality and morbidity, but that it should merely be used to identify low-risk patients and not to identify high-risk patients. Apart from this being a bit of a disappointment in itself (we are really after a score that defines high-risk), a few methodologic issues of this study and some other limitations of the GAS score need to be highlighted.

The study population that was used to validate the GAS score consisted of patients who were all considered suitable for open repair between 1994 and 2003. It is very likely that in the same period, patients who were considered at high risk for open repair (by whatever criteria) were treated by EVAR in this institution. As a result, young, low-risk patients are probably over-represented in the current study population.

Unfortunately, the authors also included juxtarenal and suprarenal AAA repairs. As this subgroup is responsible for three of the total five deaths, it obviously plays a major role in the study outcome. With only two deaths in the remaining 193 infrarenal AAA repairs, one thing is clear for these authors: they really do not need a scoring system to predict deaths, as their mortality rate of open AAA-repair is exceptionally good. The reader is left with the conclusion that GAS may be helpful in identifying low-risk patients for this infrarenal AAA population, while this seems to be based upon a study population void of high-risk patients.

Furthermore, it is important to realize that age is a major driver of the GAS score. The typical median of AAA patients at the time of surgery is between 70 and 75, which means that almost half of all AAA patients considered for repair already fall into the high-risk GAS-category (>77 points) on account of their age alone. With a history cerebrovascular or renal disease, it becomes almost impossible for a patient to qualify as low risk. Fortunately, these risk factors are uncommon (13% and 6%, respectively, in the current study). Patients with myocardial disease can only qualify as low risk if they are in their 60s. It therefore must be questioned if GAS really offers a lot more predictive power than age alone. Not to mention the issue of corrected or improved cardiac disease not being accounted for in the GAS score, as appropriately addressed by the authors.

In conclusion, the authors are to be commended for tackling a vital issue in current AAA practice. The GAS score is a clever and simple test that deserves further evaluation; however, it needs application in larger studies with a higher proportion of patients with significant risk factors.